# THE HISTOPATHOLOGIC SPECTRUM OF ALOPECIA

**DISSERTATION**

**SUBMITTED FOR**

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# CERTIFICATE

This is to certify that the dissertation work entitled **" THE HISTOPATHOLOGIC SPECTRUM OF ALOPECIA "** submitted by **Dr. Umamaheswari** is the work done by her during the period of study in this department from June 2004 to February 2007. This work has been done under my direct supervision and guidance.


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# CONTENTS

# INTRODUCTION

Hair has no vital function in man and is not required for our physical existence. It plays a role in providing protection and serves as a vehicle for the application of medications and cosmetics. In forensic medicine, the analysis of hair is useful in identification and for the detection of various poisons[22]. However, its most important function lies in the maintenance of an individual's social and psychological equilibrium[14]. Alterations in the quantity and quality of hair have a great impact on behaviour and social interactions. Diseases of the hair are not generally associated with mortality, but are responsible for significant morbidity. While most men with a family history of alopecia are able to reconcile themselves to a bald head, the situation in women is different. Hair loss in women, causes as much anguish as the growth of facial or body hair in excess of culturally acceptable norms.

About 50 to 100 hairs are shed every day from the normal scalp. When the daily hair loss exceeds 100, it is known as alopecia. Alopecia is not uncommon and the prevalence rate is 1.7% of the population. Men and women are equally affected and a family history of the disorder is present in about 25% of patients. It may be one of the manifestations of a systemic disease or a disease entity in itself. The latter consists of a variety of clinicopathologic disorders.

The distribution of hair loss and the presence of scarring are useful in making a

diagnosis. Hair loss is diffuse and uniform in telogen effluvium and patchy or patterned in alopecia areata. Scarring alopecia is characterized clinically by loss of hair follicles, which is often focal, induration or atrophy of the skin, pigmentary changes and follicular plugging. The loss of follicles is irreversible. The changes seen in non-scarring alopecia are qualitative rather than quantitative. These consist mainly of alterations in the type of hair and the growth cycle of the hair follicle.

Hair follicles are classified as terminal, indeterminate and vellus depending on the thickness of the hair shaft. They also have a distinctive morphology according to the phase of the growth cycle. These consist of actively growing/anagen, involuting /catagen and resting/telogen hair follicles. In the past, standard textbooks of histology and dermatopathology have not provided details that make these structures easy to identifyg. The most recent dermatopathology textbooks and atlases have solved the problem to a large extent [22,35]. Since scalp biopsies are infrequently received in the histopathology laboratory and conventional vertical sections have been replaced by horizontal sections it seemed necessary to study normal scalp biopsies in addition to those from patients with alopecia

# AIMS AND OBJECTIVES

1. To study scalp biopsies from normal individuals in order to be familiar with the types of hair follicles and the phases of the hair cycle

2. To assess the histological changes in the various types of alopecia.

3. To evaluate the impact of combining vertical and horizontal sections in studying alopecia

# MATERIALS AND METHODS

Scalp biopsies were received in the Histopathology department of PSG Institute of Medical Sciences and Research, Coimbatore, from 40 patients with alopecia, during the period July 2004 to February 2007. Except for 2 biopsies that were 3 mms in diameter, the rest were 4mm punch biopsies, taken from sites where the disease was active, yet not too advanced. This was usually at the periphery of the lesion. They were sent in 10% formalin and processed in an automatic tissue processor prior to paraffin embedding. Nine vertical sections were taken and mounted on 3 glass slides. The block was then melted and the tissue re-embedded with the subcutaneous tissue facing downwards and the epidermal aspect towards the technician in order to obtain horizontal sections. Serial 5µ sections were cut on a Leitz microtome with a disposable blade until the tissue was exhausted . Every $25^{th}$ to $27^{th}$ section was mounted on 3 separate glass slides and each slide had 4 levels,eg, 25,50,75,100, etc. One slide having the vertical sections and one slide each representing various levels were stained with haematoxylin-eosin(H-E). The others were stained with Verhoeff van Gieson(VVG) or Alcian blue/periodic acid-Schiff (AB/PAS) as required.

Scalp specimens, from the parieto-occipital region, taken with a 4mm punch, at autopsy, from 6 cases of road traffic accidents served as the controls.

Both control and diseased biopsies were evaluated with regard to the follicles,

stelae and dermal changes – inflammation, scarring and mucin content – as indicated in Table 1.

**Table 1.Features noted in horizontal sections**

| |
|---|
| Number of terminal follicles |
| Number of indeterminate and vellus follicles |
| Number of terminal follicles in anagen |
| Number of follicles in catagen + telogen |
| Number of follicular units |
| Number of follicular stelae |
| Follicular inflammation : location and cell type |
| Pigment casts within follicles |
| Perivascular inflammation |
| Sebaceous glands |
| Arrectores pili muscle |
| Fibrosis |
| Elastic pattern |
| Presence of mucin |

# REVIEW OF LITERATURE

The evaluation of hair loss is a diagnostic challenge to both dermatologist and pathologist. From the pathologists point of view it is necessary to have a basic knowledge of the morphogenesis, histology and physiology of the hair follicle.

**Hair follicle: morphogenesis**

The hair follicle is an epidermal appendage that together with the sebaceous gland and hair arrector muscle forms the functional unit known as the pilosebaceous apparatus. In utero, the epithelium and underlying mesenchyme interact to form the hair follicle. It is during this time that the distribution and phenotype of the hair – long hair over the scalp and short hair over the eyebrow – are determined. This is regulated by molecular signals.

At birth, the human body is covered by approximately 5 million hair follicles. Although no more follicles are formed, the follicles and hairs change with time, primarily under the influence of androgens. The distribution of follicles is determined by genes that are expressed very early in morphogenesis – lymphoid-enhancing factor 1, bone morphogenetic protein 4 and the type 11 receptor for transforming growth factor β[37]. They appear well before there is any sign of follicle formation. A little later, cells containing the protein products of the homeobox genes are seen at the exact sites where

the follicles form. These are demonstrable in the adult too, at various times of the cycle, indicating that they are necessary for both follicle formation and their sustained growth. The phenotype of hair is determined by morphogens such as sonic hedgehog, β catenin and lymphoid-enhancer factor 1 [26.]

Development and cycling of the follicles also depends on growth factors such as insulin-like growth factor 1 and fibroblast growth factor 7 which are produced in the hair papilla and interact with receptors in the matrix cells of the hair bulb. Endogenous hormones – androgens, oestrogens, thyroid hormones, glucocorticoids, prolactin and growth hormone regulate growth.The hair follicle is richly innervated and neural peptides produced at nerve endings and by Merkel cells are known to alter hair growth.

**ENDOGENOUS MODULATORS OF HAIR - FOLLICLE CYCLING IN HUMANS.[26]**

| MODULATOR | ACTION |
|---|---|
| Androgens | Promote miniaturization of follicles and shorten duration of the anagen stage in androgen -sensitive areas of scalp; enlarge follicles in androgen-dependent areas (E.g., male beard)during adolescence. |
| Estrogens | Prolong the anagen stage; postpartum reduction in estrogen secretion causes telogen effluvium |
| Growth hormone | Acts synergistically with androgen during virilization in adolescence |
| Prolactin | Can induce hirsutism |
| Thyroxine | Low levels cause telogen effluvium; high levels may have a similar effect |

**Hair follicle: biology**

There are 4 types of hair follicles:

**1. Lanugo hairs** which are present in the 7$^{th}$ and 8$^{th}$ month of foetal life. These consist of fine, soft, nonpigmented hair shafts that lack a central medulla.

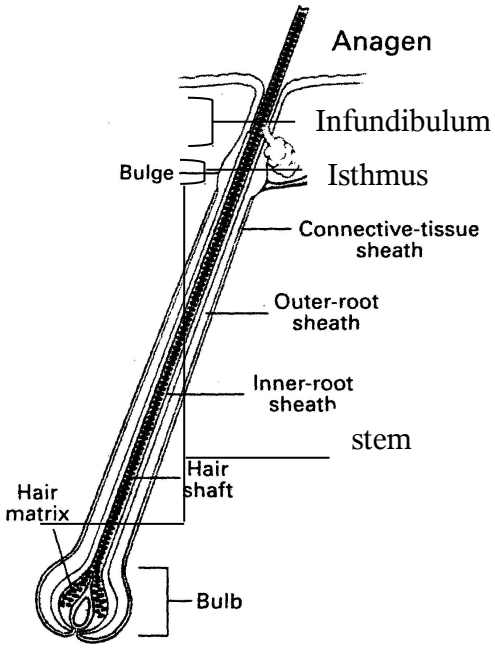
**2. Vellus hairs** are thin(less than 0.03mm diameter), short, hypopigmented with bulbs located in the upper dermis. The diameter of the hair shaft is less than that of the inner root sheath.

**3. Indeterminate hairs** are intermediate in size between terminal and vellus hairs.

**4. Terminal hairs** are thick(0.06mm diameter), long, pigmented with bulbs in the subcutaneous fat. The diameter of the hair shaft is much greater than the width of the inner root sheath

Every hair follicle regardless of its size, can be found in one of the 3 phases of the hair cycle:

**1.The anagen phase** is the actively growing period that lasts for weeks to years,depending on the site and size of the hair. For human scalp terminal hair, the anagen phase lasts between 2 and 7 years.




The anagen hair has 4 zones. From deep to superficial, these are the **bulb, suprabulbar segment or stem, isthmus and the infundibulum**. The hair bulb lies in the subcutaneous fat and consists of the matrix which is composed of rapidly proliferating cells and surrounds the dermal papilla. The papilla is continuous inferiorly with the fibrous sheath that surrounds the entire follicle. The suprabulbar segment is complex and shows differentiation into layers. From the centre to the periphery, these consist of the following layers.

i) The hair shaft medulla

ii) Hair shaft cortex

iii) The cuticular layer

iv) Huxley's layer of the inner root sheath

v) Henle's layer of the inner root sheath.

vi) The glassy (vitreous ) layer

vii) Fibrous root sheath

The isthmus lies between insertion of the arrector pili and the entry of the sebaceous duct.The inner root sheath desquamates in the midportion of the isthmus resulting in a gap between the hair shaft and the follicular wall. The epithelium adjacent to the insertion of the arrector pili is known as the bulge zone and contains the follicular stem cells. The uppermost zone is the infundibulum.This lies superior to the entry of the sebaceous duct. Some workers divide the follicle into an upper segment – isthmus and infundibulum - which is stable and unaffected by follicular maturation. The lower segment – bulb and stem – show active growth and the morphology varies with the phase of the cycle

**2.The catagen phase** is a brief, transitional phase between the anagen and telogen phases. It lasts for about 2 to 3 weeks.

The catagen follicle has an inconspicuous bulb in which the matrix cells are replaced by epithelial cells. The epithelium of the suprabulbar segment disintegrates and the papilla migrates upward into the dermis to lie at the insertion of the arrectores pili muscle. The fibrous sheath that surrounded the follicle remains below the papilla as a collapsed fibrovascular tract referred to as a stela or streamer.The epithelium above the

papilla is expanded and contains a club hair.

**3.The telogen phase** is the resting phase and lasts for about 100 days.

The telogen follicle has a hair papilla that consists of a condensed ball of spindle-shaped cells that lies just below the epithelial bulge situated at the insertion of the arrectores pili muscle. This consists of an aggregate of basaloid cells.

Depending on the individual, at any given time, 85-100% of terminal hair follicles are in the anagen phase, 0-15% in the telogen phase and about1% in the catagen phase[35].

**Specimen preparation: vertical versus horizontal sections**

Traditionally, histopathologists have relied entirely on vertical sections of most tissues, except nerve, skeletal muscle and blood vessel, for diagnostic purposes. Though vertical sections are satisfactory for the study of dermatologic diseases, their utility in the evaluation of alopecia is questionable. The hair follicle lies obliquely in relation to the epidermis. As a result of this, the follicle is often missed, cut tangentially or incomplete. **Headington**[12]noted that in a 4mm cylindrical punch of normal scalp that contains 12 to 14 follicular units only 2 to 3 follicular units were observed in vertical sections. Thus vertical sections of cylindrical punch biopsy specimens showed only 10 to 15% of follicles in the sample . The main advantage of a vertical section is that the dermoepidermal junction, papillary dermis and subcutis are better demonstrated.

Interface dermatitis, lupus panniculitis, miniaturized hairs and trichomalacia are more obvious[4].

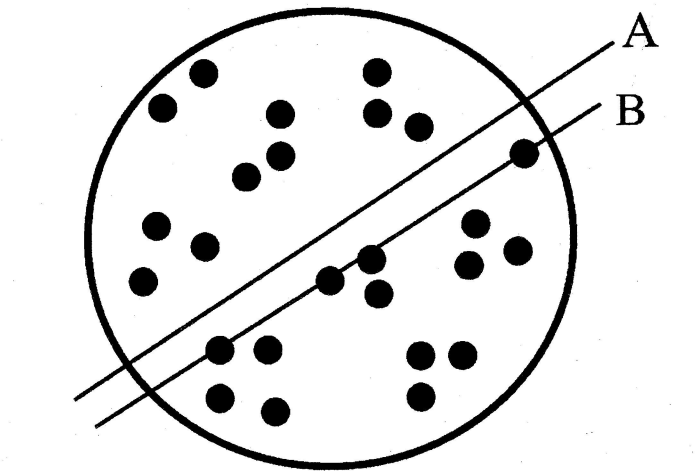


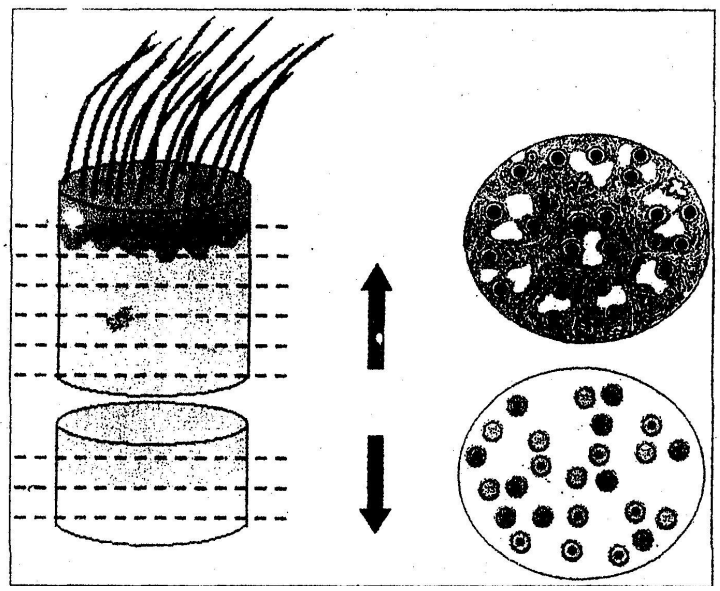

Diagram of a 4-mm punch biopsy specimen from the scalp. [37]

The advantages of transverse sections, in addition to the higher yield of follicles are the rapid, easy and accurate assessment of follicular density, follicle and shaft diameters, anagen, telogen, terminal and vellus hairs. The drawbacks are the need to use a different protocol for specimen preparation and familiarity with the transverse microanatomy of the scalp[40].

**Specimen processing for horizontal sections**

The biopsy should be fixed in 10% formalin for at least 24 hours. After this one of the following methods may be used to obtain horizontal sections:

1. A single transverse cut is made about 1mm below the epidermal surface. Both cut sides are painted with eosin and embedded down in the cassette. As the microtome cuts deeper into the block, the sections become more superficial in one half of the specimen and deeper in the other[12,42.]



Transverse sections are cut through both halves of the punch biopsy specimen. The upper half progressively displays the more superficial structures while the deeper structures are displayed in the lower half. [22]

2. The biopsy is cut transversely into 3 or 4 slices in the manner of a bread loaf. The deep surface of each slice is inked and the inked surface is placed downwards while embedding. Once the ink has been trimmed off by the microtome, a section is taken. Thus the specimen is sampled at several levels[9].

3. The entire specimen is embedded with either the epidermal or fat surface downwards and horizontal sections are taken until the block is exhausted. This is a tedious process, but has the advantage of examining the follicle thoroughly[35].

**Transverse microanatomy of the normal scalp**

It has been observed that transverse sections of the normal scalp show a constant architecture. In the superficial subcutaneous tissue and the deep dermis, the bulbs and suprabulbar segments(stems) of the terminal hair follicles are evenly spaced. However, in the mid and upper dermis the follicles are aggregated into follicular units. Morphometric studies on 4mm punch biopsies have provided numerical data that can be accepted as standard. A 4mm punch biopsy samples an area ($\pi r^2$),i.e,12.6$mm^2$.This formula can be used to compare data from punch biopsies that are larger or smaller in diameter. A 4mm punch biopsy,in a Caucasian, has about 38 hair follicles, of which 33 are terminal and 5 are vellus.. On an average, of the 33 terminal follicles, 31 are anagen and 2 are telogen. However, considerable variation occurs in the telogen count in normal individuals and figures within a range of 0-15% are acceptable. There are an average of 12 follicular units each containing 2-5 terminal hairs and 0-2 vellus hairs. The terminal:vellus ratio is 2:1 or greater[35].Studies on African Americans and Koreans show a slightly lower total number of follicles, fewer vellus hairs and a similar anagen:telogen ratio.

***Comparison of hair counts between Koreans, whites and blacks*** [22]

| | **Korean** | **Whites** | **Blacks** |
|---|---|---|---|
| Total number of cases | 35 | 22 | 32 |
| Average age (years) | 33.1±10.0 | 43 ±3.5 | 31.7±8.5 |
| Average Terminal hairs | 14.9 ±3.2 | 35 ±2.1 | 18.4 ±5.0 |
| Average vellus hairs | 1.1 ±1.3 | 5.0 ±0.6 | 3.0 ±2.1 |
| Average total hairs | 16.1±3.6 | 40± 2.2 | 21.5 ±5.0 |
| Average follicular units | 17.8 ±1.7 | 14± 0.5 | NA |
| Terminal: Vellus ratio | 13.5:1 | 7:1 | 6.1:1 |
| Anagen: telogen | 14.6:1 | 14.4:1 | 15.3:1 |
| Follicular Structures /$mm^2$ | 1.2 ±0.3 | 3.1±0.8 | 1.65 ±0.4 |

**Classification of alopecia**

Several classification schemes for alopecia have been proposed, but none of these is perfect. Most forms of alopecia demonstrate clinical and histological overlap. This blurs the distinction between diseases and makes classification difficult. The best approach would be to group diseases according to their aetiology, but this is impossible since the causes of many forms of hair loss are unknown.

The most widely accepted classification divides alopecia into **scarring** and **non-scarring** (cicatricial and non-cicatricial)forms. Scarring alopecia may be defined as an irreversible hair loss secondary to permanent destruction of the hair follicle or follicular unit. In non-scarring alopecia the hair follicle and follicular unit are intact but there is an alteration in the size and growth dynamics of the terminal follicles. Non-scarring alopecia is reversible.

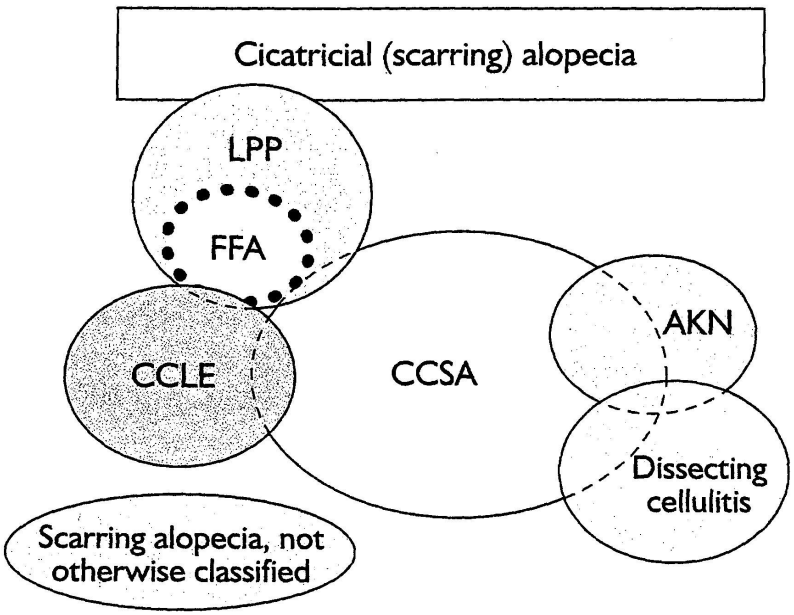
However, certain hair diseases demonstrate a **biphasic pattern**, where non-scarring hair loss is seen early in the course of the disease, and permanent hair loss becomes apparent in the later stages. Examples of these are androgenetic alopecia, alopecia areata and traction alopecia. These forms of alopecia are generally considered to be non-scarring but, after many years of continuous active disease, permanent dropout of follicles occurs [35,40].

**Scarring Alopecia**

Scarring alopecia has been subdivided into primary and secondary. In primary scarring alopecia, the hair follicle is principally involved, eg, lichen planopilaris and lupus erythematosus. In the other variant, the hair follicle is not the main target, but is incidentally involved due to surrounding dermal disease, eg, morphea, lichen sclerosis et atrophicus etc.

**Primary Scarring Alopecia**

Consists of a heterogeneous group of diseases which lack characteristic biologic markers and are therefore difficult to categorize.As such, it is necessary to acknowledge that any classification has its own problems and is in a state of evolution. At a workshop conducted by the North American Hair Research Society in 2003, a working classification based on the predominant inflammatory cellular infiltrate was developed[25]. Using this classification, Mirmarani et al found that it was not possible to differentiate the various clinical variants of scarring alopecia[21]. Hence the clinical classification continues to be used [37].




**Classification of scarring alopecia [35]**

***LPP, lichen planopilaris , ( FFA, forntal fibrosing alophecia is a subset of LPP) CCLE, chronic, cutaneous lupus erythematosus ; CCSA, central, centrifugal scarring alopecia ; AKN, acne***

***keloidalis***

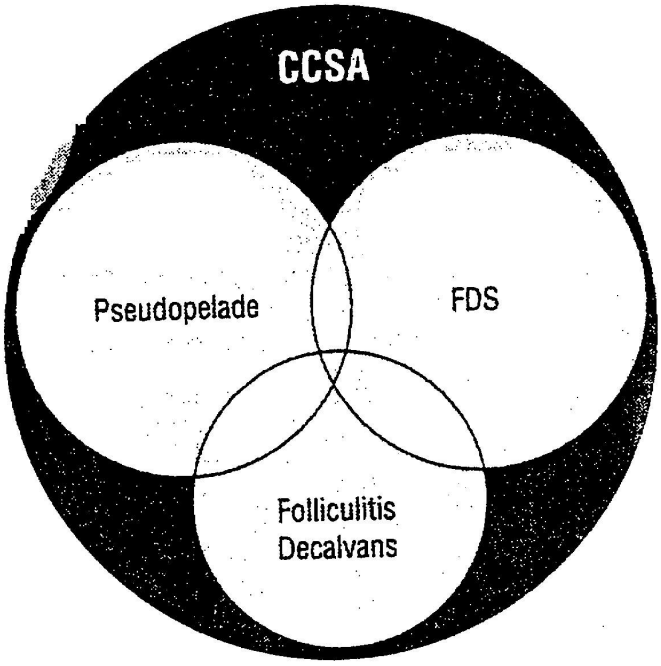
**Lichen planopilaris(LPP)**:was first described by Pringle in 1895.It is characterized by patchy hair loss with lesions of typical lichen planus elsewhere on the skin in 50% of cases. It is more frequent in women and the average age of onset is 51 years The lesions may be pruritic[22].Frontal fibrosing alopecia, described in postmenopausal women by **Kossard** in 1994 is a variant of scarring alopecia closely related to lichen planopilaris[15,16]. The triad of scarring alopecia of the scalp, loss of axillary and pubic hair with the development of keratosis pilaris, called the Graham Little syndrome was also considered to be a variant of LPP. However, typical lichen planus lesions are not seen and the histological picture is often not lichenoid [35,37].

The microscopic features consist of an interface dermatitis affecting the infundibulum and isthmus. 'Squamatization' of the basal layer, subepidermal cleavage, colloid bodies and interfollicular interface dermatitis are supportive findings. Perifollicular inflammation and fibroplasia are often the only features seen, but cannot be considered diagnostic[46.] Elastic stains show subepidermal wedge shaped fibrous tracts with loss of elastic in the upper part.Outlines of the elastic sheath of the follicles are present in the lower portion of the fibrous tract.[7] Immunofluorescence studies show staining of the colloid bodies with IgM and linear deposits of fibrin along the basal layer of the infundibulum [2,18].

**Discoid lupus erythematosus(DLE):** is the most familiar form of scarring alopecia. About 30-50% of patients with discoid lupus erythematosus present with scalp involvement. The lesions resemble those of classic DLE with alopecia, erythema, epidermal atrophy and plugged follicular ostia. Central hypopigmentation and peripheral hyperpigmentation are typical of lesions in dark skinned individuals. Plaques of DLE may coalesce and when centred on the crown or vertex resemble central, centrifugal scarring alopecia[37].

The microscopic features are those of vacuolar interface change in the epidermis and follicular epithelium, although the epidermis may be spared. Lichenoid inflammation and colloid bodies are less prominent than in LPP. When perifollicular inflammation is noted it is maximal at the infundibular level. A moderate to dense chronic inflammatory infiltrate, including plasma cells, is present around blood vessels and sweat glands [35,37,38]. Dermal mucin, oedema, telangiectasia and basement thickening favour a diagnosis of DLE. Unlike LPP, elastic stain reveals destruction of the entire length of the elastic sheath. The dermis between follicular units is also destroyed.There is scarring throughout the dermis and no fibrous tracts are seen[7]. A positive lupus band test is seen in up to 83% of cases and consists of granular deposits of IgG, C3 and C1q along the basement membrane of the epidermis and follicles.

**Central, centrifugal scarring alopecia (CCSA):** is a term that was recently coined for an inflammatory, scarring alopecia which includes the entities previously described as follicular degeneration syndrome, pseudopelade and folliculitis decalvans[3,22.,36]. It is characterized by chronic, progressive hair loss involving the crown and vertex which spreads in a roughly symmetrical fashion. There is a central zone of alopecia surrounded by active inflammation. Patients with highly inflammatory disease with intense erythema, pustules and crusting are usually African American and diagnosed as folliculitis decalvans. Those with more indolent disease in the form of perifollicular scaling with occasional papules are labeled as follicular degeneration syndrome or pseudopelade [36,37,38]




***CCSA – Central centrifugal scarring alopecia, FDS – Follicular degeneration syndrome*** [38]

The microscopic features consist of premature desquamation of the inner root

sheath and eccentric atrophy of the follicular epithelium with approximation of the hair shaft to the dermis at the site of epithelial thinning. There are perifollicular lymphocytic infiltration at the junction of the infundibulum and isthmus and concentric lamellar fibrosis. Complete destruction of the follicular epithelium and granulomatous inflammation may be seen in the advanced lesions. The pustular lesions of folliculitis decalvans show a predominantly neutrophilic perifollicular and intrafollicular infiltrate with bacteria. It is for this reason that some workers consider it an infective process that does not belong in the category of CCSA**.** [28]

**Dissecting cellulitis**: is a rare disease that predominantly affects young men of African origin. It is part of the follicular occlusion triad, but can occur in the absence of acne conglobata and hidradenitis suppurativa. Much of the scalp, particularly the crown, vertex and upper occiput are covered by boggy or fluctuant pus filled nodules. The disease varies in intensity over several years and eventually leads to dermal scarring, sinus formation, permanent alopecia and even squamous cell carcinoma.

The microscopic picture varies with the age of the lesion. The earliest change is perifollicular lymphocytic infiltration of the deep dermis, extending into the subcutaneous fat. With time, neutrophils, plasma cells and proliferating capillaries appear and the dermo-subcutis junction is converted into granulation tissue.[39] The inflammation then extends into the upper dermis, destroying follicles. Repeated injury to

the follicles leads to dilatation of the infundibulum. A notable feature is the relative sparing of sebaceous glands. [38]

**Acne keloidalis**: typically affects young African Americans and occurs as follicular papules on the occipital region and posterior neck with occasional involvement of the vertex and crown. With time, the papules heal leaving behind zones of alopecia. In many patients, they fuse to form hairless keloidal plaques. Abscesses and sinuses are present in some patients. It is frequently associated with CCSA, suggesting a common or related pathogenesis.

The microscopic features are similar to those of CCSA and consist of perifollicular lymphoplasmacytic infiltration at the junction of the isthmus and infundibulum with lamellar fibroplasia and atrophy and destruction of the follicular epithelium. [35]

**Scarring alopecia, unclassified**: is home to those cases that cannot be placed in any of the other 5 categories. Assigning poorly defined cases into this group would be better than creating a new but unproven diagnostic entity[37] . Moreover, as in all other classification schemes in pathology, the admission of ignorance is the first step to acquiring knowledge.

**Secondary scarring alopecia** occurs due to dermal disease that secondarily affects the hair follicle

## SECONDARY SCARRING ALOPECIA [40]

### Sclerosing disorders

- Morphea
- Sclerodermoid porphyria cutanea tarda
- Lichen sclerosus et atrophicus
- Perry-Romberg syndrome

### Physical/ Chemical agents

- Mechanical trauma, laceration
- Thermal burns
- Chemical burns
- Radiation dermatitis

### Dermal infiltrative processes

#### Tumors

- Basal cell carcinoma
- Squamous cell carcinoma
- Metastatic carcinoma
- Lymphoma
- Adnexal tumors
- Dermatofibrosarcoma protuberans

#### Granulomatous inflammation

- Sarcoidosis
- Necrobiosis lipoidica
- Infections

#### Amyloidosis

**Non-scarring alopecia**

The defining feature of non-scarring alopecia is that it is reversible. The total number of hair follicles is normal but there are alterations in the morphology and type of hair follicles. The changes seen are more qualitative than quantitative. However, it is recognized that some subsets of non-scarring alopecia after many years of active disease may be converted to the scarring type.

**Androgenetic alopecia(AGA):** also known as common/hereditary balding is caused by androgens in genetically susceptible males[40]. Androgens are the main regulator of hair growth. At puberty, they are responsible for the conversion of vellus to terminal hairs. However, they can have an opposite effect, leading to the replacement of terminal hairs by vellus hairs, and the onset of alopecia. This is believed to be due to increased activity of 5α-reductase type 2 present in the outer root sheath and bulb papilla of the hair follicle[1]. The enzyme converts testosterone to dihydrotestosterone which has a strong affinity for the androgen receptors in the hair follicle. The hormone receptor complex activates the genes that transform terminal to vellus hair follicles. Patients with AGA have higher levels of the enzyme and receptor in the frontal as compared to the occipital follicles. [27]

The microscopic features are miniaturization of hair follicles and an increase in the number of telogen follicles. Because of the miniaturization there is a mixture of hairs of varying size and depth of bulbs. The normal ratio of terminal:vellus hairs is between 2:1 to 7:1. This is reduced and even reversed in AGA. As hairs miniaturize, the anagen phase shortens, resulting in an increase in the telogen count [27,30]. Many of these are also miniaturized. Although the total count of follicles is normal, longstanding cases may show a reduction in the density of hair follicles – transition to scarring alopecia. Mild lymphohistiocytic infiltration is present around the infundibulum and blood vessels of the superficial dermis. The cause of this has been variously attributed to seborrheic dermatitis, actinic damage or the application of cosmetic and grooming agents [42]

**Alopecia areata(AA):**is a common form of non-scarring alopecia that affects 1% or more of the population. In 20-30% cases it is associated with other autoimmune diseases such as type1 diabetes mellitus.[10,17] It can affect any part of the body, but is most common on the scalp. Alopecia may occur as a single self limiting episode or it may recur. Typically, there is one or more round, smooth bald patches which seem depressed because of loss of the supporting effect of the hair. Short hairs that taper as they approach the scalp (exclamation hairs) are characteristic. Sometimes the entire scalp is involved – alopecia totalis; in alopecia universalis all body hair is lost [43].

The microscopic features depend upon the stage of the disease – acute, subacute or chronic. The changes of acute disease are seen in rapidly progressive disease, in early lesions and along the advancing margin of the bald patch. There is peribulbar lymphocytic infiltration around anagen bulbs and the suprabulbar segment of the hair follicle. The infiltrate may invade the dermal papilla and the matrix cells[23]. Intercellular and intracellular oedema and destruction of matrix cells and melanocytes of the hair bulb and keratinocytes of the outer root sheath can also occur. Occasional eosinophils may be present in the infiltrate.[6,19,20] It has been emphasized that peribulbar inflammation is seen only in some follicles and may even be absent. The presence of peribulbar inflammation is a helpful but not an essential diagnostic criterion. There is a marked increase in the number of categen/telogen hairs with increased stelae that may show melanin clumps and mild inflammation. The subacute stage is characterized by a marked increase in the number of catagen/telogen follicles and miniaturization of hair shafts. Inflammation is scant and the residual anagen follicles may show thinning of the hair shafts. The chronic stage shows numerous miniaturized hair follicles as well as catagen/telogen follicles. In addition there are nanogen follicles that are characterized by a thin outer root sheath, central cornification and no hair shaft. Inflammation is mild.

***Telogen effluvium:*** occurs when abnormally large numbers of anagen follicles, from all parts of the scalp, progress to catagen and subsequently telogen. The cause of this may be physiological as in the newborn and postpartum. Pathological causes include severe acute and chronic illness, surgery, thyroid disorders and drugs. Some postmenopausal women suffer from a chronic telogen effluvium that lasts more than 3 months, has no known cause and runs a fluctuating course.

The microscopic changes are subtle and consist of increased numbers of telogen follicles and stelae. The percentage of telogen follicles is often greater than 20%. This figure is arbitrary and lower counts may sometimes be encountered in patients with chronic disease. There is no inflammation or change in the terminal:vellus ratio [40].

***Trichotillomania:*** is a form of traumatic alopecia caused by traction and plucking of hair. It usually represents a chronic mental illness, but can be seen in healthy persons who have gone through significant stress. There are irregular, sharply demarcated bald patches in which a few hairs of various lengths are present.

The microscopic features are quite distinctive and consist of distortion of follicular anatomy. There is no hair shaft and the inner root sheath is collapsed. When the inner root sheath has also been removed the outer root sheath is distorted. Sometimes the epithelium is almost totally removed and there is red cell extravasation. Pigment casts may be present within the

follicle. There is a significant increase in the catagen/telogen count secondary to trauma . The mechanical injury precipitates anagen arrest.[11,31]

# RESULTS

## CONTROL SPECIMENS :

These consisted of 6 punch biopsies of the scalp, taken from the parieto-occipital region of 4 females and 2 males between 18 and 55 years of age.

Microscopically,they showed occasional bulbs and stelae in the subcutaneous fat (Fig 1,2).The stem and upper segment of the terminal anagen follicles appeared normal (Fig 3 )Terminal anagen follicles were evenly distributed at the dermo-subcutaneous junction and grouped together to form follicular units in the mid and upper dermis (Fig 4,5 ). The average total number of follicles was 26.2 with the terminal:vellus ratio of 9.3:1 and the anagen:telogen ratio of 8.9:1. Like the vellus follicles, catagen and telogen follicles were present in the upper and mid dermis (Fig 6,7,8). A mild lymphohistiocytic infiltrate was present around the upper segment of hair follicles in one specimen and around blood vessels of the superficial dermis in all specimens.

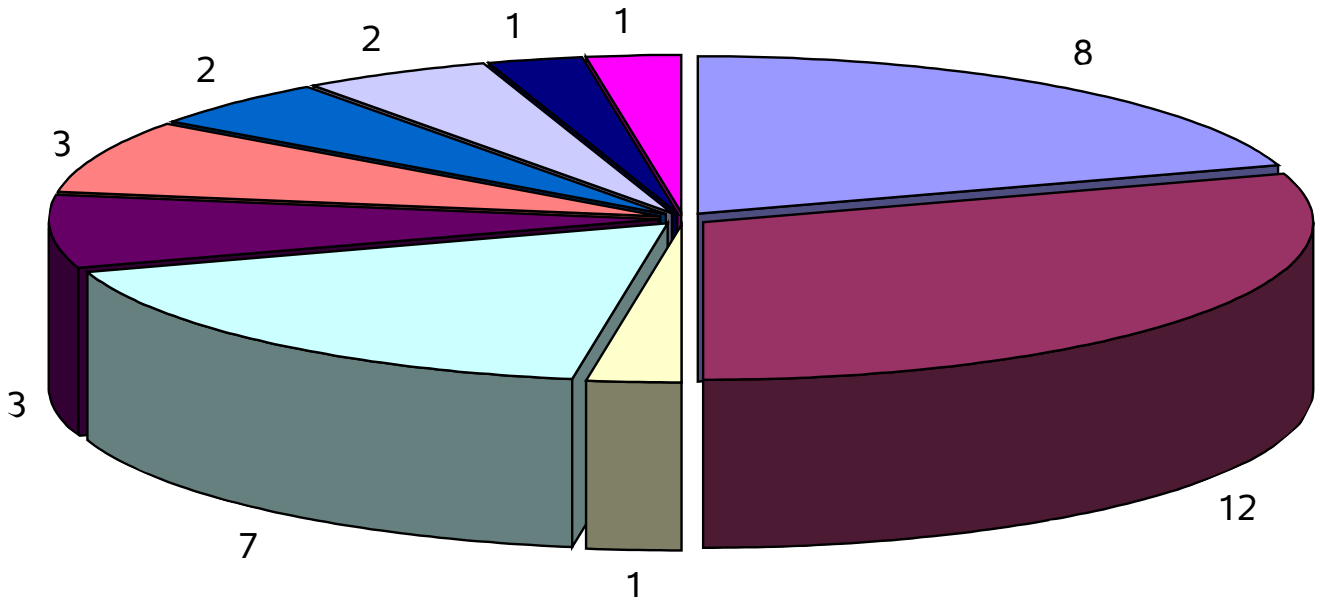
**TABLE 1 – MICROSCOPIC FEATURES IN CONTROL SPECIMENS**

| Control No | Number of | | | | Terminal : Vellus Ratio | Anagen : Telogen Ratio | Perifollicular inflammation | | Perivascular inflammation | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Bulbs | Stelae | Follicles | Follicular units | | | Upper | Lower | Superficial | Deep |
| 1 | 1 | 1 | 19 | 6 | 2.8:1 | 6:1 | - | - | + | - |
| 2 | 1 | 0 | 30 | 10 | 9:1 | 8:1 | - | - | + | - |
| 3 | 0 | 0 | 30 | 10 | 13:1 | 14:1 | - | - | + | - |
| 4 | 2 | 3 | 23 | 12 | 23:0 | 3.6:1 | + | - | + | - |
| 5 | 0 | 2 | 24 | 7 | 3.8:1 | 18:1 | - | - | + | - |
| 6 | 0 | 1 | 31 | 12 | 4.2:1 | 4:1 | - | - | + | - |

## Alopecia Specimens

There were 19 cases of scarring alopecia and 21 cases of non-scarring alopecia. Among the 19 cases of scarring alopecia there were 7 cases of lichen planopilaris and 3 cases each of discoid lupus erythematosus and unclassified scarring alopecia. Two cases each of scleroderma and end-stage scarring alopecia and one each of lichen sclerosis et atrophicus and atypical necrobiosis lipoidica were diagnosed. The group of non-scarring alopecia consisted of alopecia areata 12 cases, androgenetic alopecia 8 cases and trichotillomania 1 case.

## INCIDENCE OF ALOPECIAS IN SCALP BIOPSY SPECIMENS



**Lichen planopilaris (n=7)**

There were 4 males and 3 females between the ages of 23 and 37 years. Pruritus and lesions typical of lichen planopilaris were present elsewhere on the body in 2 cases. All cases had been diagnosed clinically as lichen planopilaris, but in 2 cases an alternative diagnosis of alopecia areata had been offered.

Microscopically, lymphocytic infiltration of the basal layer,( lichenoid inflammation,) of the upper segment of the follicle was present in 6 biopsies (fig 9). The interfollicular epidermis showed lichenoid inflammation in 2 biopsies. All biopsies showed perifollicular concentric fibrosis (fig 10). Atrophic, angulated follicles were seen in 5 biopsies and the epithelial atrophy was eccentric in 4 (fig11). The upper dermis showed pigment incontinence in all cases.The Verhoeff van Gieson stain showed wedge shaped subepidermal fibrous scars and in some of these, the outline of the elastic sheath of the follicle was seen in the lower portion of the fibrous tract.(Figs12,13) Follicular units were not identified in 3 biopsies. Lymphocytes were present around blood vessels of the upper dermis. Mucin was noted around follicles in 4 biopsies. Hyperkeratosis with hypergranulosis was seen in the epidermis in 2 biopsies.

## TABLE – 2 MICROSCOPIC FEATURES OF LICHEN PLANOPILARIS

| Biopsy no | Lichenoid inflammation | | Perifollicular Concentric Fibrosis | Dermal inflammation | | Dermal Mucin | | Pigment incontinence | Follicles Units | Follicles | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Epidermal | Follicular | | Superficial | Deep | Diffuse (D) | Perifollicular(F ) | | | Eccentric Atrophy | Angulated Contour |
| 1399/04 | - | - | + | + | - | - | + | + | 3 | + | + |
| 1841/04 | + | + | + | + | - | - | + | + | 5 | + | + |
| 2329/05 | - | + | + | + | - | - | - | + | 5 | - | + |
| 624/05 | + | + | + | + | - | - | + | + | 6 | - | + |
| 2137/05 | - | + | + | + | - | - | - | + | 0 | - | - |
| 1889/06 | - | + | + | + | - | - | - | + | 0 | + | - |
| 2932/06 | - | + | + | + | - | - | + | + | 0 | + | + |

**Discoid lupus erythematosus(n=3)**

All patients were female between 26 to 45 years of age. In 2 cases, the lesions were pruritic and accompanied by other lesions on the skin of the face. These were clinically compatible with discoid lupus erythematosus.

Microscopically, perifollicular, lichenoid lymphocytic infiltration affecting the upper segment was present in all biopsies. Involvement of the epidermis was present in 1 biopsy. Melanophages were seen in the upper dermis in all biopsies and there was thickening of the epidermal basement membrane in one biopsy (fig 14,15). There was full thickness scarring of the dermis with loss of elastic pattern and absence of follicular units in all biopsies. The follicles were atrophic and atrophic as well as angulated in 1 biopsy each. Inflammation, both superficial and deep was present around blood vessels in all, and was perieccrine in one biopsy (fig 16). Plasma cells were present in the inflammatory infiltrate in one biopsy. Dermal mucin was abundant and diffuse in all biopsies (fig 17).

**TABLE – 3 MICROSCOPIC FEATURES OF DISCOID LUPUS ERYTHMATOUS**

| Biopsy no | Lichenoid inflammation | | Perifollicular Concentric Fibrosis | Dermal inflammation | | Dermal Mucin | | Pigment incontinence | Follicles Units | Follicles | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Epidermal | Follicular | | Superficial | Deep | Diffuse (D) | (F)Perifollicular | | | Eccentric Atrophy | Angulated Contour |
| 785/05 | - | + | - | + | + | D | - | + | 0 | - | - |
| 802/05 | + | + | - | + | + | D | - | + | 0 | - | + |
| 1103/06 | - | + | - | + | + | D | - | + | 0 | - | + |

**TABLE – 4 MICROSCOPIC FEATURES OF UNCLASSIFIED SCARRING A LOPECIA**

| Biopsy no | Lichenoid inflammation | | Perifollicular Concentric Fibrosis | Dermal inflammation | | Dermal Mucin | | Pigment incontinence | Follicles Units | Follicles | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | Epidermal | Follicular | | Superficial | Deep | Diffuse (D) | (F)Perifollicular | | | Eccentric Atrophy | Angulated Contour |
| 2577/05 | + | + | + | + | + | + | - | + | 10 | + | + |
| 1975/06 | + | + | + | + | + | + | - | _ | 6 | + | + |
| 229/05 | - | + | + | + | - | - | - | - | 6 | - | - |

**Unclassified scarring alopecia (n=3):**

A 44 year old male, had a depigmented atrophic plaque on the vertex and was clinically diagnosed to have discoid lupus erythematosus.A 20 year old female with a pruritic pigmented lesion in the frontal region was clinically diagnosed to have lichen planopilaris.Both cases showed lichenoid inflammation of the epidermis and the upper segment of the follicles. Both biopsies also showed concentric perifollicular fibrosis.There was atrophy of the follicles, angularity and eccentric epithelial atrophy. Inflammation was present throughout the dermis and there was a diffuse increase in dermal mucin. Verhoeff van Gieson stain showed wedge shaped subepidermal fibrous scars in the upper and mid dermis.

The third case was a 55 year old man who presented with diffuse hair loss, pruritus and prominent follicles.A clinical diagnosis of diffuse alopecia areata was made. Microscopically the dominant finding was the presence of irregular masses of amorphous black material with surrounding foreign body reaction (fig 18). There was miniaturization of hair follicles and lichenoid inflammation around follicles in the upper dermis.

**End-stage scarring alopecia(n=2)**

A 15 year old female presented with bald patches of 5 years duration. Microscopically only 3 terminal anagen and 1 catagen follicles were present in the entire biopsy. The anagen follicles were atrophic and in one of them the epithelial atrophy was eccentric There was concentric fibrosis around the follicles. There was lymphocytic infiltration around blood vessels in the upper dermis and the sebaceous glands were atrophic. Many stelae were present. The elastic stain showed the outlines of 6 fibrosed follicular units.

A 35 year old man presented with itching of the scalp and diffuse hair loss. Microscopically there were no hair follicles or sebaceous glands. Stelae were increased and there was pigment within these as well as in the upper dermis. Lymphocytic infiltration was present throughout the dermis and the elastic stain showed 4 fibrosed follicular units. There was mild increase in dermal mucin.

**Scleroderma (n=2)**

A connective tissue disorder was clinically diagnosed in 2 females aged 18 and 37 years, with patchy hair loss over the scalp.

Microscopically, both biopsies showed homogenization of the upper dermis with loss of elastic fibres. The rest of the dermis was rather acellular with broad collagen fibres. One case showed epidermal atrophy and marked reduction of hair follicles, all of which were telogen follicles. The subcutaneous fat showed necrosis and lobular lymphocytic panniculitis and no hair bulbs were seen. The other biopsy showed normal subcutis. The hair bulbs and follicles were surrounded by a thick sheath of hyalinized fibrous tissue with increase in vellus and telogen hair follicles – terminal:vellus 2:1, anagen:telogen 3:1 (Fig 19)

**Lichen sclerosis et atrophicus(n=1)**

A 35 year old female presented with hair loss over medial aspect of left eye brow and scalp of 6 years duration. A clinical diagnosis of linear morphea/ alopecia areata was made.

Microscopically there was hyperkeratosis, epidermal atrophy & acanthosis with homogenization of collagen and melanin incontinenece in the upper dermis. There was vascularity and perivascular inflammation at the junction of the upper and mid dermis.The bulbs in the subcutaneous tissue were normal. There were 5 anagen follicles with mild atrophy, angularity of the outline and one of these showed eccentric epithelial atrophy. No arrectores pili or sebaceous glands were seen.

**Necrobiosis lipoidica(n=1)**

A 40 year old non-diabetic female with a depigmented atrophic, hairless lesion in the frontal scalp of 11 months duration was diagnosed to have vitiligo/ lichen sclerosis et atrophicus/ linear morphea.

Microscopically there was epidermal thinning with foci of necrosis and loss of elastic in the upper dermis. They were surrounded by a histiocytic infiltrate ( Fig. 20 ). No mucin was present. There was intimal thickening of arteries and a perivascular lymphoplasmacytic infiltrate throughout the dermis. Four anagen follicle with perifollicular fibrosis and a telogen follicle were seen.

**Alopecia areata (n=12):**

There were 11 female and one male patient in the age range of 2 to 53 years.A diagnosis of alopecia areata was made in 8 cases and alopecia universalis in 2 cases.Microscopically a peribulbar lymphocytic infiltrate was present in 9 biopsies (fig 23). Eosinophil infiltration and miniaturization were present in 2 biopsies. Stelae were increased and contained clumps of melanin.There was a marked increase in telogen follicles.In 9 biopsies there were follicles that were neither typically anagen or telogen - nanogen follicles.The ones that resembled anagen follicles had a central solid collection of amorphous keratin and the hair shaft was minute or absent (fig24,25).Those follicles that resembled catagen follicles appeared larger with a convoluted rather than angulated outline.

## TABLE - 5 - MICROSCOPIC FEATURES OF ALOPECIA AREATA

**Androgenetic alopecia (n=8):**

There were 6 males and 1 female, aged between 21 and 40 years with hair loss over the frontal region and crown.

Microscopically, there was miniaturization of hair follicles that were typically of varying diameter(fig21).The hair shafts too were thinner and the average terminal:vellus ratios was 1:8.Telogen follicles were increased and the average anagen:telogen ratio was 5:1.Mild perivascular lymphocytic infiltration was present in the upper dermis in all biopsies and mild perifollicular lymphocytic infiltration of the upper segment in 5.The hair bulbs were normal and there were many stelae. One case also showed premature desquamation of the inner root sheath and eccentric atrophy of the follicular epithelium (fig 22).

There was an 18 month old female with diffuse hair loss of 3 months duration.The microscopic features were similar to the other cases with terminal:vellus ratio of 1:4 and anagen:telogen ratio of 5;1.

## TABLE- 6 - MICROSCOPIC FEATURES OF ANDROGENETIC ALOPECIA

**Trichotillomania**

A 23 year old female presented with patchy hair loss, pigmentation, broken hairs and follicular prominence of 5 years duration.

Microscopically there was distortion of follicles with loss of epithelium and collapse of the inner root sheath. Pigment casts were present in the keratin overlying the epidermis and within the follicles ( Fig 26,27)

# DISCUSSION

Histopathologic examination is routinely done for the diagnosis of many dermatologic diseases. Although hair loss is distressing and not infrequent, scalp biopsies form a minor component of specimens received in the histopathology laboratory. For decades, the microscopic examination of hairs that have been pulled, plucked or spontaneously shed have been used for the evaluation of hair loss. This is useful in diseases of the hair shaft and to an extent in non-scarring alopecia, but of limited value in most cases of alopecia. Attempts to study the histological changes in alopecia, by conventional vertical sections, were disappointing as no useful information was forthcoming and no clinical correlates could be established. Both dermatologists and pathologists groped for a diagnostic approach to the problem of alopecia.

In 1984, Headington[12] described and illustrated the morphology of hair follicles in transverse sections of the scalp. He indicated that the morphometric analysis of transverse sections would provide information that could be used to study hair follicles in health, disease and assess response to therapy. Subsequent workers have shown that horizontal scalp sections are the preferred method for evaluating all forms of hair loss [4,9,33,41,43].

When the study commenced , we were unfamiliar with the transverse microanatomy of the scalp in health and disease. The illustrations and text in standard books of dermatopathology provided little information on this[5,8,42]. The study of control specimens obtained at autopsy familiarized us with the structure and architecture of the follicular components of the scalp. Since we are accustomed to vertical sections we thought it would be better to have a few vertical sections, in all biopsies for orientation. This was found to be particularly useful in the detection of interface dermatitis in primary scarring alopecia and for demonstrating the subepidermal wedge-shaped scars of lichen planopilaris. Elston et al have also demonstrated that combined vertical and horizontal sections increase the diagnostic yield[4].

From the study of control specimens we determined the total number of hairs,terminal:vellus ratio and anagen:catagen ratios.This data is helpful in interpreting scalp biopsies as there is an ethnic variation in hair follicle counts.Since the control group consisted of only six specimens and there were none from healthy volunteers,with no history of hair loss, we plan to obtain more accurate statistics in a subsequent study.

**Primary scarring alopecia**

Lichen planopilaris was the commonest type of primary scarring alopecia. There was no gender predisposition and lesions of typical lichen planus elsewhere on the body were infrequent. Pigment incontinence, indicative of interface dermatitis was seen in all biopsies. Lichenoid inflammation was present in all, except one. This inflammation was always follicular and rarely involved the interfollicular epidermis. Concentric perifollicular fibrosis was seen in all biopsies. Perifollicular mucin was a frequent finding but diffuse dermal mucin deposition as in discoid lupus erythematosus was not seen. Scarred follicular units (blank spots) were also noted. Atrophic follicles and eccentric epithelial atrophy which has been regarded as typical of central,centrifugal scarring alopecia were also present.

Discoid lupus erythematosus was less frequent in this series and all patients were female. Like lichen planopilaris there was melanin incontinence and lichenoid inflammation of the follicle in all cases, with epidermal involvement in only one case. Diffuse deposition of dermal mucin, diffuse dermal scarring with loss of the elastic pattern and inflammation of the deep dermis were seen in all cases.

There were 2 biopsies where it was not possible to differentiate whether scarring alopecia was secondary to lichen planopilaris or discoid lupus erythematosus. These were placed in the group of unclassified scarring alopecia. Both biopsies showed lichenoid follicular inflammation, subepidermal wedge shaped scars and concentric perifollicular fibrosis. However, in one biopsy there was diffuse deposition of dermal mucin while the other showed perieccrine inflammation. These 2 biopsies underscore the need to perform direct immunofluorescence study in all cases of scarring alopecia.

A third case in this group showed perifollicular lichenoid inflammation and fibrosis with miniaturization of hair follicles and many stelae. There was foreign body giant cell reaction to black, homogeneous material. The miniaturization of follicles suggested androgenetic alopecia but the origin of the black pigment was obscure. The patient was lost to follow up and the cause of scarring could not be elucidated.

There were 2 cases where the disease was advanced. The hair follicles were absent or sparse with absence or atrophy of sebaceous glands. The inflammation was sparse and scarring was extensive. These were labeled as end-stage scarring alopecia.

In this subset of primary scarring alopecia there were no cases of central ,centrifugal scarring alopecia. This is not entirely unexpected because it is most frequently seen in African Americans. But, eccentric epithelial atrophy of follicles which is an important feature of central,centrifugal scarring alopecia was seen in biopsies of lichen planopilaris, discoid lupus erythematosus and end-stage scarring alopecia.It is interesting that premature desquamation of the inner root sheath which is considered to be diagnostic of central centrifugal scarring alopecia was seen in one case of androgenetic alopecia[13].

**Secondary Scarring Alopecia-**

The diagnosis of scarring alopecia is easily made on vertical sections. However , the horizontal section may provide an insight into the mechanism of hair loss. One case of scleroderma with lymphocytic panniculitis showed only telogen follicles suggesting that inflammation of the adipose tissue caused injury to the hair bulbs and propelled them into the telogen phase. The rest showed anagen follicles that were probably distorted or destroyed by the surrounding dermal changes.

**Non-scarring Alopecia-**

There is a histological overlap between androgenetic alopecia and alopecia areata since both are related to abnormalities in the growth cycling of hair follicles. Miniaturization of hairs and catagen / telogen transformation are seen. Peribulbar inflammation is absent in androgenetic alopecia and present, but not always in alopecia areata. In this study it was observed that 2 of the 3 biopsises of alopecia areata that lacked peribulbar inflammation showed large number of nanogen hair follicles and this was helpful diagnostically. No nanogen follicles are seen in androgenetic alopecia. The third case showed only telogen follicles.

Unusual features noted in the group of androgenetic alopecia were its occurrence in an 18 months old female. The youngest case described in literature was 6 years old

No case of telogen effluvium was present in this series. This is not unusual since patients with telogen effluvium are rarely biopsied. There was only one case of trichotillomania and this was typical, both clinically and histologically.

Cosmetology is a rapidly progressing branch of dermatology.The solution to alopecia is not a wig. Treatment strategies to control and reverse hair loss are being developed.Male androgenetic alopecia responds to topical

minoxidil and oral finasteride that are inhibitors of 5 alpha reductase[35]. Immunomodulatory drugs such as tacrolimus and cyclosporin A, as well as biologic agents that target cell surface receptors eg etanercept, infliximab etc are being considered as therapeutic options for androgenetic alopecia[2729,34]. Studies on the bulge zone of hair follicles, which is the repository of multipotent stem cells that support hair cycling, are under way[24.]. This raises the hope for gene therapy in alopecia, in the future. It cannot be overemphasized that the effect of any form of therapy is essentially dependent on an accurate histopathologic diagnosis.

# SUMMARY AND CONCLUSIONS

This study was done on scalp specimens from 6 autopsies and 40 cases of alopecia.

The study of scalp specimens from medico - legal autopsies provided useful information concerning the terminal : vellus and anagen : catagen ratios, in Indians. This data, which is considered helpful for diagnosis of androgenetic alopecia and alopecia areata, has not been reported in the Indian population. However , it needs to be validated by a study on a larger sample taken from healthy volunteers.

Scarring and non-scarring alopecia were almost equally represented in this series.

Lichen planopilaris was the commonest variant of scarring alopecia. It was possible to distinguish it from discoid lupus erythematosus by the absence of inflammation in the deep dermis, diffuse deposition of dermal mucin and extensive dermal scarring. But, in 2 cases there was an overlap of microscopic features and this underscores the need for immunofluorescence studies in cases of scarring alopecia.

There were no cases of central, centrifugal scarring alopecia which is characterized by eccentric atrophy of the follicular epithelium and premature desquamation of the inner root sheath. This was seen in cases of scarring alopecia and in one case of androgenetic alopecia respectively, which raises the issue that these features may not be specific to central, centrifugal scarring alopecia .

Unusual forms of secondary scarring alopecia- scleroderma, lichen sclerosus et atrophicus and necrobiosis lipoidica were also studied.

Alopecia areata was the commonest cause of hair loss and was diagnosed on the presence of peribulbar inflammation and / or nanogen hair follicles.

Although it is cumbersome, a more complete and thorough examination is possible by combining vertical and transverse sectioning of the same punch biopsy.

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# MASTER CHART

| S.No | Biospy Number | Name | Age & sex | Op.No | Diagnosis |
|---|---|---|---|---|---|
| 1. | S1399/04 | Sivalingam | 37/M | 004031779 | Scarring Alopecia - LPP |
| 2. | S1473/04 | Akila | 23/F | 04/33460 | Trichotillomania |
| 3. | S1841/04 | Palaniswamy | 38/M | 04/40860 | Scarring Alopecia - LPP |
| 4. | S1943/04 | Poongodi | 41/F | 04/43136 | Alopecia Areata |
| 5. | S2329/04 | Satishkumar | 35/M | 04/35789 | Scarring Alopecia - LPP |
| 6. | S2532/04 | Kavitha | 35/F | OSR | Scarring Alopecia - LSEA |
| 7. | S229/05 | Palaniappan | 53/M | 05/5493 | Alopecia - unclassified / scarring in an AGA |
| 8. | S453/05 | Padmavathy | 53/F | - | Alopecia Areata |
| 9. | S624/05 | Gunasekaran | 23/M | 005012861 | Scarring Alopecia - LPP |
| 10. | S785/05 | Ilamathy | 42/F | 005002837 | Scarring Alopecia - DLE |
| 11. | S746/05 | Joshi s.parmer | 18/F | 005015045 | Scarring Alopecia - Scleroderma |
| 12. | S997/05 | Akshayalakshmi | 11/2/F | 05/19837 | Androgenetic Alopecia |
| 13. | S1311/05 | Mani | 40/F | 05/25782 | Alopecia Areata |
| 14. | S1632/05 | Subbulakshmi | 40/F | OSR | Scarring Alopecia - necrobiosis lipoidica |
| 15. | Case 9 | Gangadharan | 26/M | OSR | Androgenetic Alopecia |
| 16. | Case 10 | Muthu | 25/M | OSR | Alopecia Areata |
| 17. | Case 11 | Kulanadaivelu | 30/M | OSR | Androgenetic Alopecia |

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| 18. | Case 12 | Ramakrishnan | 40/M | OSR | Androgenetic Alopecia |
| 19. | Case 13 | Maruthathal | 46/F | OSR | Alopecia Areata |
| 20. | Case 14 | Sirajudheen | 22/M | OSR | Androgenetic Alopecia |
| 21. | Case 15 | Mohan | 21/M | OSR | Androgenetic Alopecia |
| 22. | Case 16 | Krishna kumar | 32/M | OSR | Androgenetic Alopecia |
| 23. | Case 17 | Karthika | 11/F | OSR | Alopecia Areata |
| 24. | Case 18 | Nandhini | 10/F | OSR | Alopecia Areata |
| 25. | Case 19 | Karthi | 2/F | OSR | Alopecia Areata |
| 26. | Case20 | Tamilselvi | 15/F | OSR | Alopecia Areata |
| 27. | Case 21 | Thaslinma | 15/F | OSR | Endstage scarring alopecia |
| 28. | S2137/05 | Kavithasatish | 30/F | 05/42454 | Scarring Alopecia - LPP |
| 29. | S54/06 | Fathima ravi | 27/F | - | Androgenetic Alopecia with premature desquamation |
| 30. | S783/06 | Nagamani | 37/F | 06/6620 | Scarring Alopecia - Scleroderma |
| 31. | S802/06 | Sasilekha | 26/F | 06/15591 | Scarring Alopecia - DLE |
| 32. | S1103B/06 | Rajeswari | 45/F | 06020975 | Scarring Alopecia - DLE |
| 33. | S1290/06 | Rabiya | 30/F | 06/24359 | Alopecia Areata |
| 34. | S1144B06 | Kannammal | 41/F | 06/9953 | Alopecia Areata |
| 35. | S1889/06 | Saradhamani | 34/F | 06/16061 | Scarring Alopecia - LPP |
| 36. | S1975/06 | Venkatachalam | 46/M | 06/37448 | Scarring Alopecia - LPP Vs DLE |
| 37. | S2577/05 | Revathy | 20/F | - | Scarring Alopecia - LPP Vs DLE |
| 38. | S564/06 | Selvakumar | 33/M | 04054347 | Endstage scarring alopecia |
| 39. | S2599/06 | Thilaka | 48/F | 05/25859 | Alopecia Areata |
| 40. | S2932/06 | Rukiya | 35/F | OSR | Scarring Alopecia - |

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